Circadian dynamics in measures of cortical excitation and inhibition balance

Abbreviated title: Circadian excitation/inhibition balance

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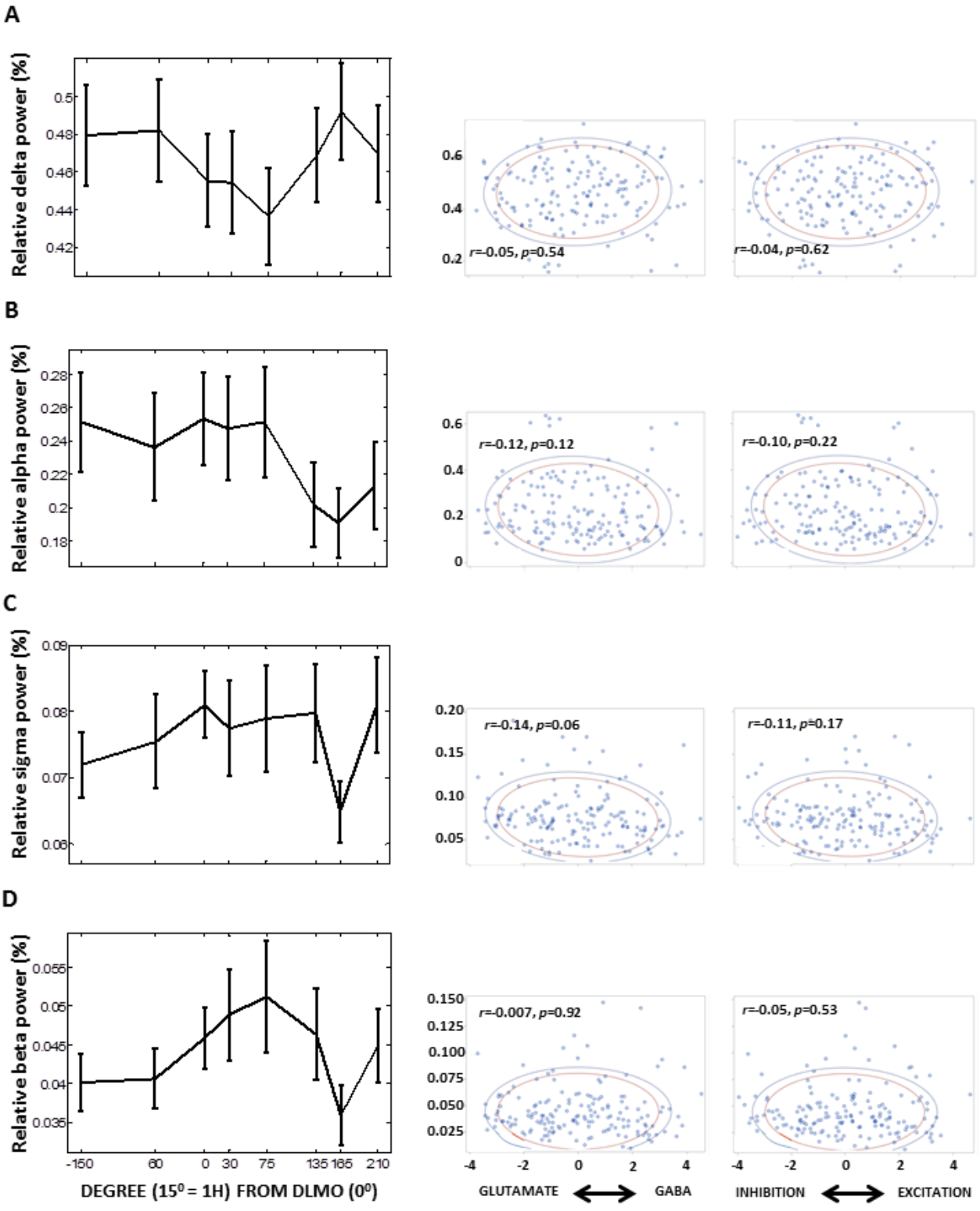

A
Relative delta power (%)
0.5
0.48
0.46
0.44
0.42
0.6
0.4
0.2
r=-0.05, p=0.54
r=-0.04, p=0.62
B
Relative alpha power (%)
0.28
0.26
0.24
0.22
0.2
0.18
0.6
0.4
0.2
0
r=-0.12, p=0.12
r=-0.10, p=0.22
C
Relative sigma power (%)
0.09
0.08
0.07
0.06
0.20
0.15
0.10
0.05
r=-0.14, p=0.06
r=-0.11, p=0.17
D
Relative beta power (%)
0.055
0.05
0.045
0.04
0.035
0.150
0.125
0.100
0.075
0.050
0.025
r=-0.007, p=0.92
r=-0.05, p=0.53
-150
60
0
30
75
135
165
210
DEGREE (15⁰ = 1H) FROM DLMO (0⁰)
-4
-2
0
2
4
GLUTAMATE
GABA
INHIBITION
EXCITATION

Figure S1. Changes in frontal EEG power across 29h of sustained wakefulness were not correlated with DCM indices of excitation/inhibition balance.

On all plots n = 22, and data are realigned according to individual melatonin secretion onset (phase 0°). Horizontal axis corresponds to time in degrees (15° = 1h) relative to melatonin secretion onset.

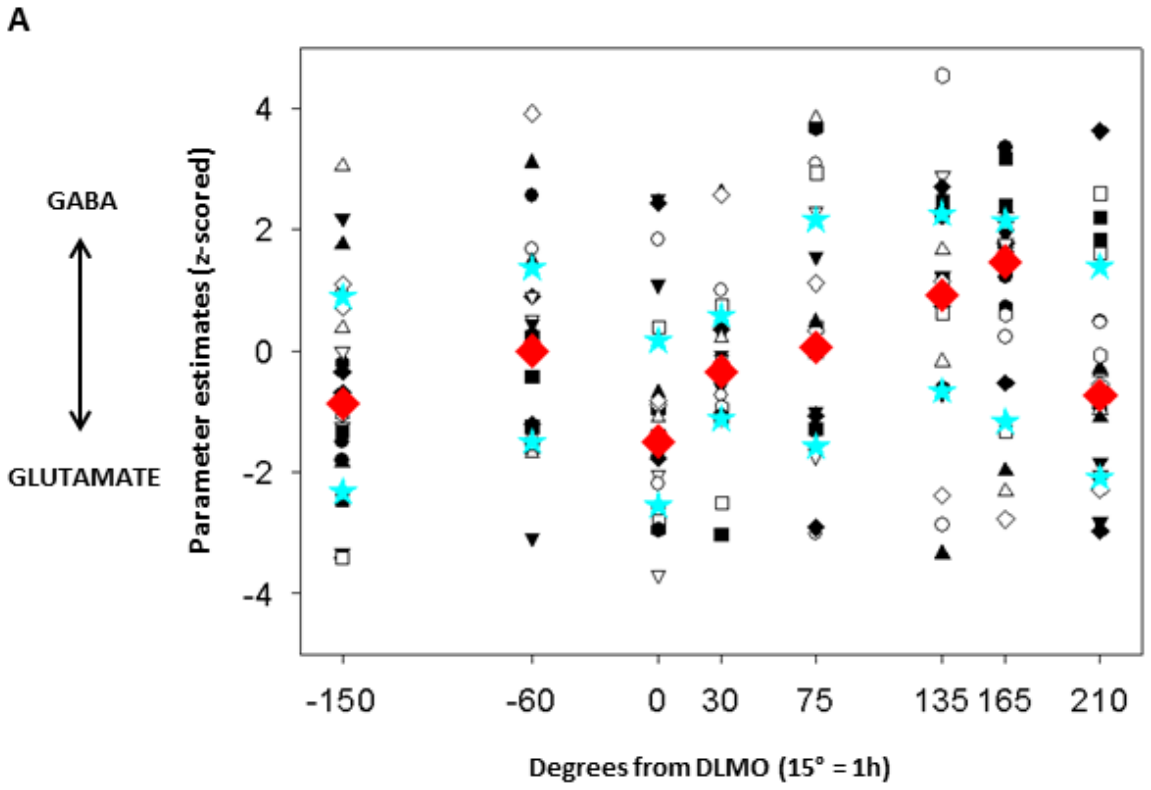
A. Relative delta power (0.75-4Hz) did not change significantly across the protocol (main effect of circadian phase: $F_{7,109}$= 1.53, p = 0.17) and was not correlated to GABA/Glutamate receptor density balance (Spearman correlations; r = 0.05, p = 0.54; middle panel) and cell-to-cell excitation/inhibition connectivity balance (Spearman correlations; r = 0.04, p = 0.62; right panel).

B. Relative alpha power (8-12Hz) changed significantly across the protocol (main effect of circadian phase: **$F_{7,109}$= 3.79, p = 0.001**) but was not correlated to GABA/Glutamate receptor density balance (Spearman correlations; r = -0.12, p = 0.12; middle panel) and cell-to-cell excitation/inhibition connectivity balance (Spearman correlations; r = -0.1, p < 0.22; right panel).

C. Relative sigma power (12.5-18Hz) did not change significantly across the protocol (main effect of circadian phase: $F_{7,111}$= 1.73, p = 0.11) and was not correlated to GABA/Glutamate receptor density balance (Spearman correlations; r = -0.14, p = 0.06; middle panel) and cell-to-cell excitation/inhibition connectivity balance (Spearman correlations; r = -0.11, p = 0.17; right panel).

D. Relative beta power (18.5-30Hz) did not change significantly across the protocol (main effect of circadian phase: $F_{7,112}$= 1.84, p = 0.09) and was not correlated to GABA/Glutamate receptor density balance (Spearman correlations; r = -0.007, p = 0.92; middle panel) and cell-to-cell

excitation/inhibition connectivity balance (Spearman correlations; r = -0.05, p = 0.53; right panel).


A
GABA
GLUTAMATE
Parameter estimates (z-scored)
4
2
0
-2
-4
-150
-60
0
30
75
135
165
210
Degrees from DLMO (15° = 1h)

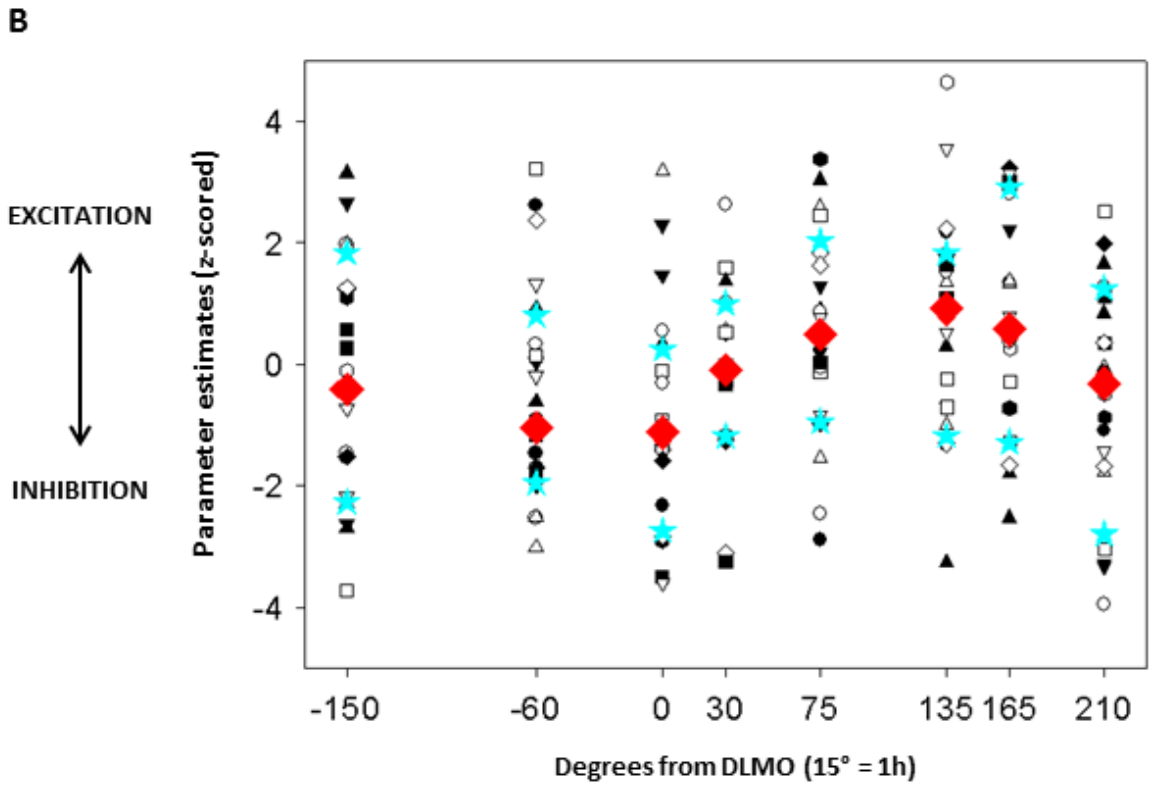

B
EXCITATION
INHIBITION
Parameter estimates (z-scored)
4
2
0
-2
-4
-150
-60
0
30
75
135
165
210
Degrees from DLMO (15° = 1h)

Figure S2. Individual changes in GABA/Glutamate receptor time constant and cell-to-cell excitation/inhibition connectivity balances during normal waking and sleep deprivation (related to figure 3).

On both plots n = 22, and data are realigned according to individual melatonin secretion onset (phase 0°). Horizontal axis corresponds to time in degrees (15° = 1h) relative to melatonin secretion onset.

Red diamonds: median values; **Blue starss**: 20% and 80% percentiles

A. GABA/Glutamate receptor time constant balance variations (z-scored parameters at the individual level, as on figure 3).

B. Excitation/inhibition cell-to-cell connectivity parameter balance variations (z-scored parameters at the individual level, as on figure 3)

Overall both plots indicate that there is inter-individual variability in the variations of both indices. Therefore, in addition to the significant changes reported in the main text which we relate to changes in sleep homeostasis and circadian phase, there are other sources of variations in the indices of excitation and inhibition we computed. These interindividual variations should be investigated in future experiments.